

**A STUDY ON CLINICAL MANIFESTATIONS  
OF RIGHT VENTRICULAR  
MYOCARDIAL INFARCTION**

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## **CERTIFICATE**

This is to certify that this dissertation entitled “**A STUDY ON CLINICAL MANIFESTATIONS OF RIGHT VENTRICULAR MYOCARDIAL INFARCTION**” submitted by **Dr. MAGESH. V** appearing for Part II M.D. Branch I General Medicine Degree examination in March 2009 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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## **DECLARATION**

I solemnly declare that the dissertation titled “**A STUDY ON CLINICAL MANIFESTATIONS OF RIGHT VENTRICULAR MYOCARDIAL INFARCTION**” is done by me at Madras Medical College & Govt. General Hospital, Chennai during 2006-2009 under the guidance and supervision of **Prof. M. JUBILEE , M.D.**

The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfilment of requirements for the award of M.D. Degree (Branch I) in General Medicine.

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# INTRODUCTION

Myocardial Infarction is the term used when the myocardium is necrosed due to ischemia. It may be transmural or subendocardial.

Inferior wall infarction has got some special features like association with Right ventricular infarction and Brady arrhythmias especially sinus bradycardia and heart blocks.

Right Ventricular Myocardial Infarction is different from that of the left ventricle in the acute presentation, therapy and long term prognosis. The early recognition of Right Ventricular Myocardial Infarction is important, because the time of onset of its haemodynamic consequence is unpredictable and these may be prevented by the administration of intravenous fluid load.

Right Ventricular Myocardial Infarction( RVTMI) was first reported as a separate clinico pathological entity in 1930 by Sandas. In their review of 2000 consecutive necropsies in 1948, Wastman and Hellerstein<sup>1</sup> described 22 instances of Right Ventricular Myocardial Infarction out of 164 cases of acute Myocardial Infarction. Wade et al<sup>2</sup> noted at necropsy, the major damage in Right Ventricular Myocardial Infarction was to the inferior wall.

Right Ventricular Myocardial Infarction was described as a separate clinical entity by Cohn et al<sup>3</sup> in 1974.

The diagnosis of Right Ventricular Myocardial Infarction became practical and easy when Erhardt et al<sup>4</sup> introduced right precordial electrocardiography.

Isolated Right Ventricular Myocardial Infarction is very rare. It is usually associated with Inferior Wall Myocardial Infarction. It is less commonly associated with Anteroseptal Myocardial Infarction.

The incidence of Right Ventricular Myocardial Infarction in Inferior wall infarction varies from 25-52 % in various studies<sup>3-10</sup>.

Involvement of right ventricle is related to severe atherosclerotic occlusion of the right coronary artery and is associated with involvement of postero inferior wall and posterior portion of septum.

Clinically Right Ventricular Myocardial Infarction can be suspected when a patient with Inferior Wall Myocardial Infarction presents with elevated JVP, positive Kussmaul's sign, hypotension, right sided third or fourth heart sounds, tender hepatomegaly, oliguria rarely TR and clear chest.

There are various electrocardiographic criteria to diagnose Right Ventricular Myocardial Infarction<sup>11,12,13</sup>. Chou et al<sup>14</sup> proposed that ST elevation in V1 might suggest Right Ventricular Myocardial Infarction. Candell<sup>15</sup> et al described correlation between ST elevation in V4R and hemodynamic and scintigraphic findings for Right Ventricular Myocardial Infarction.

Introduction of hemodynamic studies, radionuclide ventriculography, pyrophosphate scintigraphy, phonocardiography and 2D echocardiography have made the diagnosis of subclinical cases easier and earlier<sup>16,17,18</sup>.

In this study clinical manifestations of Right Ventricular Myocardial Infarction are studied based on electrocardiographic criteria.



# **REVIEW OF LITERATURE**

## **Historical Aspects**

The word 'chest pain' is the vague and older terminology which includes organic and psychogenic causes of varied etiology. The term 'anginal pain' was first described by Herberden<sup>19</sup> in 1768.

The electrocardiographic studies on human heart, was first introduced by Waller<sup>20</sup> in 1887. Einthoven<sup>21</sup> elaborated the electrocardiographic study and published Einthoven's law of triangle in 1912. Wilson et al<sup>22</sup> introduced unipolar limb and chest leads in ECG.

Modern era said to have begun with the autopsy studies of Herrick<sup>23</sup> in the year 1912. He concluded that the clinical syndrome of Myocardial Infarction result from acute thrombotic occlusion of coronary arteries, with resulting downstream necrosis.

Right Ventricular Myocardial Infarction was identified as a unique clinico pathological and hemodynamic entity by Cohn<sup>3</sup> and his associates in 1974. This ushered a new era in the diagnosis and management of Right Ventricular Myocardial Infarction.

## **Coronary Circulation**

The heart is supplied by two coronary arteries namely the left and right coronary artery. Left main coronary artery arises from the left coronary cusp and divides into left anterior descending coronary artery and left circumflex coronary artery.

Right coronary artery arises from the right coronary cusp of aorta and passes forward and to the right to emerge between the root of the pulmonary trunk and right atrial appendage. It then descends in the right atrio-ventricular groove, curves posteriorly at the acute margin of the right ventricle to reach as far as the posterior interventricular groove. It ends by anastomosing with left circumflex artery.

Right coronary artery gives the posterior interventricular branch (or) posterior descending branch which runs along the posterior interventricular groove and ends by anastomosing with left anterior descending artery near the apex of the heart.

Right coronary artery supplies the posterior one third of the interventricular septum and the adjacent part of both ventricles. It also supplies A-V node, proximal His bundle and posteromedial papillary muscle. The right coronary artery also gives many

branches to supply the free right ventricular wall. It supplies the sinus node in 60% of population.

So, if there is a proximal obstruction of right coronary artery there is infarction of right ventricle along with infarction of the inferior wall of the left ventricle, whereas if the obstruction is distal to the branches supplying right ventricle, there is infarction of inferior wall of left ventricle only.

### **Variations:**

1. The two coronary arteries may arise separately (or) from the same sinus.
2. Sometimes 3 or 4 coronary arteries may be present.
3. Right coronary branch, conus artery which is usually its first branch (64%) but sometimes arises separately in the anterior sinus as a third coronary artery.
4. Left coronary opening may be double.

❖ Right dominance means the posterior interventricular artery arise from right coronary artery 90%.

❖ Left dominance means the posterior interventricular artery arise from left coronary artery 10%.

In chronic coronary obstruction and ischemia collateral connection develops via subendocardial, subepicardial, transseptal and transatrial vascular connections and the vascular beds become variably interdependent.

## **Epidemiology**

Acute Myocardial Infarction continues to be a major public health problem in both developed and developing countries, despite impressive strides in the diagnosis and management.

Although acute myocardial infarction death rate has declined by above 30% over last decade, it is still a fatal event in approximately one third of patients.

In our country

- ❖ While coronary artery disease had been halved in west in the past 50 years the rates have been doubled in India<sup>24</sup>

- ❖ Coronary artery disease prevalence in Urban areas–10%  
Rural areas – 15 %<sup>25-27</sup>

## **Peculiarities of Indian Coronary artery diseases:**

1. Extreme prematurity, coronary artery disease mortality in an Indian <30 years of age is 3 fold higher than whites<sup>28</sup>

2. Women have rates similar to men despite smoking being uncommon in Indian women<sup>29</sup>
3. Higher prevalence of glucose intolerance and lower prevalence of conventional coronary risk factors<sup>30</sup>

Although atherosclerosis is the most common cause of luminal narrowing in coronary artery disease, multiple non-atherosclerotic causes account for 4-7% cases of acute myocardial infarction<sup>31,32</sup>

## **Pathogenesis and Pathology**

Coronary atherosclerosis in the form of stenosing plaques is the major etiological factor behind the various clinical syndromes of ischemic heart disease. In a small minority, non atherosclerotic causes like emboli, vasculitis and dissection may be seen.

Stenosis caused by atheromatous plaque may be eccentric or concentric<sup>33</sup>. Eccentric plaque causes total variation in lumen size leading to variable luminal stenosis. Concentric plaque has fixed stenosis<sup>34</sup>.

Fatty streaks - which are the earliest lesion in atherosclerosis starts in coronaries of children, by 10 years. With increasing age, fatty streaks evolve into fibromuscular plaques<sup>35</sup>.

Platelet adherence, platelet aggregation and release of smooth muscle growth factors cause the embryonic atherosclerotic plaque to increase in their size.

Smooth muscle and fibrous tissue growth factors are released from disturbed endothelium and macrophages. Abnormal lipid transport from arterial lumen followed by necrosis and calcification occurs in atherosclerotic plaque.

Infarction occurs when an atherosclerotic plaque fissures, rupture or ulcerate and when condition favour thrombogenesis, so that mural thrombus forms at the site of rupture and lead to coronary occlusion.

Atherosclerotic plaque can be classified according to morphology into stable and unstable plaque<sup>36</sup>.

### **Mechanism of thrombosis**

Three forms of vascular injury were proposed by Ip et al<sup>37</sup>

Type I – Endothelial dysfunction

Type II – Endothelial denudation and intimal damage with intact internal elastic lamina

Type III - Endothelial denudation and damage of both intima and media

Rupture of plaque is a function of internal plaque changes and external stress.

Internal – Plaque vulnerability

External – Systemic triggers – Blood pressure changes,

- Cigarette smoking, Cold exposure

- Exercise, emotions, erect posture

- Local triggers – Infection, Inflammation<sup>34</sup>

Platelet activation with release of thromboxane  $A_2$  is an accompanying phenomenon – Hirsch et al.<sup>38</sup>

It promotes conformational change in glycoprotein II b/ III a receptor leading to platelet aggregation. Then the coagulation cascade activated leading to conversion of prothrombin to thrombin which then converts fibrinogen to fibrin. Ultimately coronary artery becomes occluded by a thrombus containing platelet aggregates and fibrin strands.

### **Risk factors for Atherosclerosis / Acute Myocardial Infarction<sup>39</sup>**

Category – I

#### Non modifiable risk factors

- Age

- Male gender
- Family history of early onset coronary artery disease
- Low socio economic status

#### Modified risk factors

- ↑ Low density lipoprotein
- ↓ High density lipoproteins
- Atherogenic diet
- Cigarette smoking
- Hypertension
- Left ventricular Hypertrophy
- Thrombogenic factor

#### Category II

- Diabetes mellitus
- Physical inactivity
- Elevated triglycerides
- Obesity

#### Category III

- Lipoprotein(a)
- Homocysteine
- Inflammatory markers - particularly C reactive protein



- Postmenopausal status

In India<sup>40</sup>

- High prevalence of diabetes , insulin resistance syndrome and central obesity
- Lower prevalence of conventional risk factors like hypertension, obesity, cigarette smoking and high cholesterol
- Emerging newer risk factors like high lipoprotein (a) homocysteine, apo B, triglycerides, fibrinogen, platelet activating factor inhibitor - I
- Low levels of high density lipoprotein and high density lipoprotein 2b
- Small dense low density lipoprotein

### **Circadian variation**

Acute Myocardial Infarction has marked circadian periodicity with peak prevalence between 6 A.M. and noon. There is 3 fold increase in frequency of infarction at around 9 A.M.<sup>41</sup>. This is due to diurnal variation in thrombotic tendencies like platelet aggregability<sup>42</sup>, circadian variation in blood pressure<sup>43</sup> and sympathetic nervous system<sup>44</sup>.

## **Right Ventricular Infarctions are less common – why?**

Right ventricular myocardial infarction occurs less commonly than would be expected from the frequency of atherosclerotic lesions involving the right coronary artery. Various reasons have been offered to explain this disparity<sup>9,45-50</sup>.

- a. The thinness of right ventricular wall allows some nutrition to be derived directly from the blood within right ventricular cavity.
- b. The intercoronary collateral system of the right ventricle is richer than that of the left ventricle.
- c. Oxygen demand of the right ventricle is low.
- d. Some parts of the right ventricle are directly nourished by thebesian vessels.
- e. Collateral flow to the ventricle is both systolic and diastolic, whereas it is mainly diastolic for the left ventricle.
- f. Energy requirement per gram of functioning myocardium is lower for the right ventricle. So the same degree of ischemia is better tolerated by the right ventricle without getting infarcted.

## **Collateral circulation**

An important collateral to the posterior circulation is via the Kugel's arteria anastomotica. It may arise from the proximal right coronary artery or the left main coronary artery and runs from anterior to posterior along the base of the atrial septum. Helen, Grover et al<sup>51</sup> have suggested that moderator band artery (which is up to 1 mm in diameter) may provide a major source of collateral flow through the anterior papillary muscle of the right ventricle.

Wade<sup>2</sup> emphasized that in cases of right coronary occlusion, right ventricle receives much of its blood supply through the left coronary artery, particularly through branches that traverse the septum and anterior one third of right ventricle. He claimed that, though right ventricular infarction occurs as a result of right coronary occlusion, preexisting lesions in the left anterior descending artery plays an important role in the pathogenesis.

The anastomosis between the fine branches of right coronary artery are poorly developed at birth and they progressively increase as age advances. This involves the newly formed fine vessels in the epicardial fat (rami telae adipose). However these

are not usually adequate enough to maintain an efficient collateral circulation if coronary artery branch is obstructed.

### **Isolated Right Ventricular Infarction**

Isolated right ventricular infarction is very rare and has been reported in the recent past in a few studies.<sup>52-57</sup> In the most cases it was due to the occlusion of a right marginal artery (or) a small right coronary artery which vascularise only the right ventricle.

Colantorio et al<sup>47</sup> have postulated that in a few of their cases, right ventricular infarction might have been secondary to acute or chronic pulmonary hypertension especially in the presence of right ventricular hypertrophy with normal or stenotic coronary arteries.

Right atrial infarction was noticed along with right ventricular infarction in 14-35% of cases. The greater incidence of right atrial involvement is explained by the fact that right coronary artery is the most important source of blood to the right atrium.

Sometimes isolated right ventricular myocardial infarction may mimic additional inferoposterior left ventricular infarction

on surface electrocardiography as reported by Mittal<sup>52</sup> et al. This probably might have been due to a dilated right ventricle.

### **Hemodynamic changes in RVM**

The hemodynamic changes observed in right ventricular myocardial infarction are secondary to right ventricular dysfunction.

The salient features include

- a) Right sided filling pressure greater than left sided filling pressure
- b) An inspiratory increase in mean right atrial pressure and right ventricular end diastolic pressure
- c) Normal or mildly increased left ventricular filling pressure
- d) Normal systolic pressure in the right ventricle and pulmonary artery

The hemodynamic criteria required to diagnose right ventricular myocardial infarction is that the right atrial (or) right ventricular end diastolic pressure must be equal to or greater than a simultaneously measured left ventricular end diastolic pressure or pulmonary capillary wedge pressure, provided the right atrial pressure is  $> 10$  mm Hg. If right atrial pressure is less than 10 mm Hg<sup>58</sup>, a volume loading test is done following which the pressures are measured again.

These changes result from decrease in the compliance of right ventricle and possibly also due to the resistance offered by the pericardium to the dilating heart. A severe non compliant pattern of right ventricle is defined as Y descent deeper than X descent in right atrial pressure tracing. These changes are responsible for right ventricular  $S_3$ ,  $S_4$ . Right ventricular infarct may mimic constrictive pericarditis, when there is equilibration of the diastolic pressure of both sides, a diastolic dip and plateau is present in both right and left pressure tracing and there is no inspiratory fall in the right atrial pressure. These hemodynamic features of right ventricular myocardial infarction may not be seen in the presence of hypovolemia.

### **Low output syndrome in RVMI**

The low output syndrome in right ventricular myocardial infarction is attributed to inadequate left ventricular filling. When right ventricular contractions are impaired severely as in right ventricular myocardial infarction, the filling of the left ventricle is entirely dependent on the distension of the right ventricle and right atrium by any of the following mechanisms<sup>59</sup>.

- a) An increase in right ventricular end diastolic volume which could augment the contractile force of the residual functioning fibres of the right ventricle
- b) Increased right atrial contractile force which might help to pump the blood into the pulmonary artery in the absence of right ventricular contraction
- c) Increased passive blood flow across the right to left atrial pressure gradient, through the low resistance pulmonary vascular bed.

Right ventricular ischemia increases the left ventricular chamber stiffness with shift of the interventricular septum to the left, which is enhanced by the restrictive effects of the pericardium. This also contributes as to the low output state in right ventricular myocardial infarction.

### **History, Physical examination and Management of Acute Myocardial Infarction**

The classical symptom of acute myocardial infarction is Chest discomfort which is retrosternal or pericardial described as pressure, aching, burning, crushing, squeezing, heaviness, sweating or bursting in quality. It is usually radiated to medial aspect of arm, neck or jaw, back etc<sup>60</sup>.

Approximately 23 % of acute myocardial infarction has the following atypical clinical features

- ❖ Nausea or vomiting/ indigestion ( common with inferior wall myocardial infarction)
- ❖ A typical location of pain-arm, back,jaw, occiput
- ❖ Profound fatigue of rapid onset
- ❖ Pulmonary edema of sudden onset
- ❖ Mental obtundation
- ❖ Severe ventricular dysrhythmias
- ❖ Confusion, vertigo, syncope
- ❖ Cerebral or peripheral embolus

The older the patient, the more atypical symptoms manifest<sup>61</sup>. Painless myocardial infarction is common in elderly and diabetics due to autonomic dysfunction

## **Examination - features of Inferior Myocardial**

### **Infarction**

#### **General Examination**

- ❖ Patient is anxious and restless
- ❖ Pallor with respiration and coolness of the extremities
- ❖ Temperature elevation upto 38° celcius during the first week



## **Vital signs**

The acute inferior wall myocardial infarction is very often associated with transient hypotension and sinus bradycardia due to Bezold Jarisch reflex. This is the reflex response of bradycardia and hypotension due to the stimulation of inhibitory cardiac receptors in the infero posterior wall of left ventricle. About half of inferior wall infarction shows evidence of parasympathetic hyperactivity

## **Rhythm of pulse**

Inferior wall myocardial infarction frequently produces sinus node and AV node dysfunction manifested as irregularities in pulse.

Pulsus paradoxus occurs if right ventricular myocardial infarction complicates inferior wall myocardial infarction

## **JVP**

Right ventricular involvement in inferior wall myocardial infarction is associated with JVP changes

- Elevated JVP
- Prominent 'a' waves
- Kussmaul's sign

AV dissociation associated with inferior wall myocardial infarction produces cannon a waves

## Precordial palpation

- Precordium is usually quiet
- Diffuse apical impulse
- Dyskinesias – paradoxical bulge in late systole
- Palpable S<sub>4</sub>
- Paradoxical splitting rarely due to severely left ventricular dysfunction

## Auscultation

- Muffled heart sounds
- Muffled S<sub>1</sub> common in acute inferior wall myocardial infarction due to prolonged PR interval
- S<sub>4</sub> - 98% cases of acute myocardial infarction<sup>62</sup>
- S<sub>3</sub> - 15-20 % cases of acute myocardial infarction<sup>63</sup>
- Pericardial rub
- Crescendo – decrescendo mid systolic murmur due to ischemia of papillary muscles
- Mitral regurgitation murmur
- Pan systolic murmur due to Ventricular Septal rupture

**Examination of Lung fields** : Pulmonary congestion occurs in 30-40% cases of uncomplicated myocardial infarction

## **Killip and Kimball Classification**

- Class – I : No pulmonary rales or S<sub>3</sub>
- Class – II : Bibasilar rales that persist after coughing (< 50 % lung fields) or S<sub>3</sub>
- Class – III : Rales over one - half of the lung fields bilaterally with radiographic evidence for pulmonary edema ( > 50 % of lung fields)
- Class – IV : Cardiogenic Shock

## **Clinical features of Right Ventricular**

### **Myocardial Infarctions**

Patients with right ventricular myocardial infarction will show clinical features right ventricular dysfunction only when the infarction is haemodynamically significant

Salient clinical features of a dominant right ventricular myocardial infarction can be summarised as below

**Symptoms** - The patients will present with retrosternal chest pain associated with sweating, nausea and vomiting. Giddiness and syncopal attacks may be present. Dyspnoea is usually uncommon

**Signs** - Systemic hypotension will be present often associated with peripheral vasoconstriction. Pulsus alternans and pulsus paradoxus may be present depending on the severity of the right ventricular myocardial infarction. Mean JVP is usually elevated and kussmaul's sign may be present. Patient may have oliguria. Auscultation of the heart may reveal a right ventricular  $S_3$ ,  $S_4$  and a pansystolic murmur of tricuspid regurgitation. Lungs are usually clear without any evidence of pulmonary vascular congestion. Liver may be palpable and tender.

**Kussmaul's Sign** – Inspiration is a simple bedside method to stress the right ventricle. This increases the venous return to the heart. A normal healthy right ventricle can cope up with this increased inflow facilitated by the reduced pulmonary vascular resistance during inspiration. A failing right ventricle will not be able to cope up with this increased inflow. This is reflected as an increased right ventricular end diastolic pressure with which is transmitted to the right atrium and hence to the jugular veins. As a result mean JVP increases during inspiration, which is called as Kussmaul's sign.

**Pulsus paradoxus** - with extensive damage to the right ventricle associated with inferior wall infarction, pulsus paradoxus has been reported. This has to be differentiated from pericardial effusion and cardiac tamponade.

Constrictive pericarditis, restrictive cardiomyopathy, cardiac tamponade and pulmonary embolism should be considered in the differential diagnosis of right ventricular infarction.

**Clinical triad**

Distended neck veins

Clear lung fields

Hypotension

**Electrocardiographic changes**

Standard 12 lead ECG plus right ventricular lead and posterior chest leads, (15 lead ECG) are always necessary to diagnose right ventricular myocardial infarction.

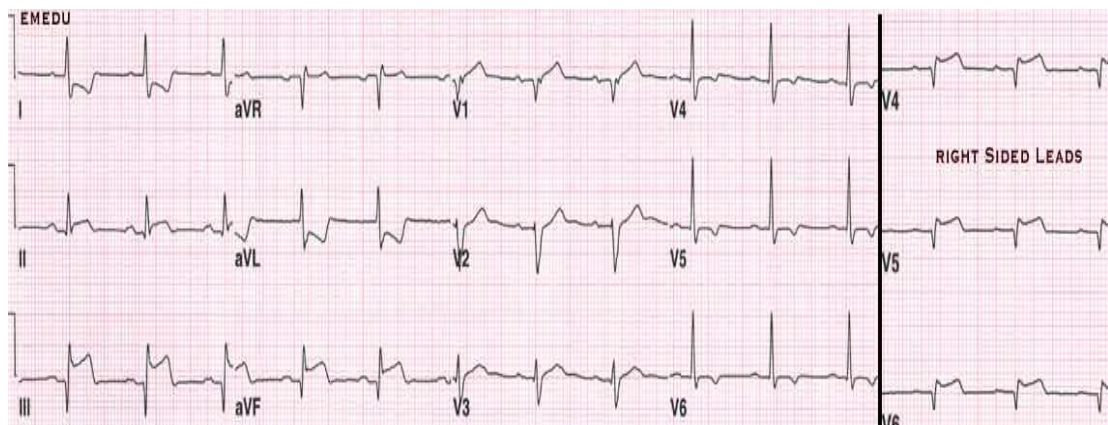
ST segment elevation greater than 1mm in right chest leads, especially V<sub>4</sub>R is essential to diagnose right ventricular myocardial infarction<sup>56,64-71</sup>.

Presence of Qs complex in V<sub>4</sub>R, ST V<sub>2</sub>/ ST aVF ratio less than 0.5, ST elevation in V1 with diminishing ST

elevation in  $V_2$  and  $V_3$  are also supposed to indicate right ventricular myocardial infarction.

Failure of reciprocal ST segment depression to develop in the right precordial leads in the case of inferior wall infarction is also significant.

Lewis<sup>70</sup> and associates reported the hyperacute phase of right ventricular myocardial infarction. This is manifested as slope elevation of ST segment in leads  $V_1$  and  $V_4R$  or only in lead  $V_4R$ .



Occlusion of right coronary artery, proximal to right ventricular branch is diagnosed by changes in  $V_4R$ .

ST segment elevation  $V_4R$  with a positive 'T' wave predicts proximal right coronary artery occlusion.

Absence of ST elevation with positive 'T' wave predicts a distal right coronary artery occlusion.

Presence of ST segment depression in anterior leads points to right coronary artery occlusion.

### **Markers used in the Diagnosis of Acute Myocardial Infarction**

S. No	Marker	Time of initial elevation (Hrs)	Mean time to elevation (Hrs)	Time to return to normal range	Most common sampling schedule
1	Troponin-I	3-12	24	5-10 days	Once atleast 12 hrs after chest pain
2	Troponin-T	3-12	12-48	5-14 days	Once atleast 12 hrs after chest pain
3	CK-MB	3-12	24	2-3 days	Every 12 hrs x 3 days
4	Myoglobin	1-4	6-7	24 hours	Frequently 1-2 hour chest pain
5	LDH	10	24-48	10-14 days	Once atleast 12 hrs after chest pain

### **Enzymatic criteria for diagnosis of Myocardial Infarction<sup>72</sup>**

- ❖ 15 % of CK in the myocardium is CK- MB, which provides sensitivity and specificity for its diagnostic marker of acute myocardial infarction
- ❖ Serial increase, then decrease of plasma CK-MB, with a change > 25 % between any 2 values

- ❖ CK-MB > 10-13 u/L or more than 5% of total CK activity
- ❖ CK-MB / CK activity more than 2.5 indicates myocardial rather than skeletal muscle as a source of CK-MB
- ❖ Increase in CK-MB activity > 50% between any 2 samples, separated by atleast 4 hours
- ❖ If only a single sample available, CK-MB elevation > 2 fold
- ❖ Beyond 72 hours, an elevation of Troponin T or I or LDH1 > LDH 2

**Early diagnosis** of myocardial infarction is offered by CK-MB subforms and Myoglobin of which CK-MB subforms are most specific.

- ❖ Absolute level of CK-MB 2 isoform greater than 1.0 u/Lt within 4-6 hrs of myocardial infarction.  
(OR)
- ❖ CK-MB 2/ CK-MB 1 greater than 2.5 has sensitivity & specificity around 90%<sup>73</sup>.



## **Troponins**

- ❖ Cardiac specific troponins which are highly sensitive and specific manner
- ❖ They increase after Myocardial Infarction to the level over 20 times higher than the cut-off value
- ❖ Levels of cTroponin-I may remain elevated for 7-10 days and cTroponin-T may remain elevated for upto 10-14 days

## **Myoglobin**

- ❖ First cardiac marker that rises above normal range after acute myocardial infarction
- ❖ Lacks cardiac specificity and levels return to normal range within 24 hours of the onset of infarction

## **Diagnosis of re-infarction<sup>74</sup>**

- ❖ Serum cardiac marker that remains elevated in the blood briefly such as CK-MB or Myoglobin is useful.
- ❖ Reinfarction defined as the secondary elevation of CK-MB activity, 36-48 hrs after onset symptoms and the value should be more than 50% above the preceding baseline.

## **Other Biochemical alterations**

- ❖ Serum cholesterol and lipoprotein fractions are relatively unchanged in the initial 1-2 days but decrease significantly over subsequent days and weeks. Hence lipid measurements are done in 24-48 hours or 6-8 weeks later<sup>75</sup>.
- ❖ White blood cell count show mild to moderate elevation in 3-5 days.
- ❖ ESR raises slowly and remains elevated for 1-2 weeks.

## **Imaging Techniques**

### **Chest roentgenogram**

- ❖ The chest film is used to exclude other causes of chest pain such as pneumothorax, pulmonary infarction with effusion, aortic dissection, skeletal fracture and so on.
- ❖ It is also used to evaluate cardiac size, the presence of pulmonary edema and atherosclerotic changes<sup>76</sup>.

### **Echocardiography**

- ❖ Early detection of the presence or absence of wall motion abnormalities can aid in the management.

- ❖ Estimation of left ventricular function by echocardiogram is useful prognostically<sup>77</sup>.
- ❖ Useful in the diagnosis of right ventricular infarction, ventricular aneurysm, pericardial effusion and left ventricular thrombus<sup>78</sup>.
- ❖ Doppler echocardiography is useful in the diagnosis of ventricular septal defect and acute mitral regurgitation.

### **Radionuclide Assay**

#### **❖ Technetium 99m sestamibi scan**

It has potential use in identifying infarct in patients with atypical presentations or in patients with ECGs that are not interpretable. Normal findings are associated with extremely low risk of subsequent cardiac events.

#### **❖ Thallium scanning<sup>79</sup>**

Thallium accumulates in the viable myocardium.

- Perfusion imaging has been used in risk stratification after myocardial infarction and for measurement of infarct size to evaluate reperfusion therapies.
- Recent advances include dual-score 64-slice CT scanning that can do a full scan in 10 seconds and produce high resolution images that allow

fine details of the patient's coronary arteries to be seen. This technology allows for noninvasive and early diagnosis of coronary artery disease and thus earlier treatment before the coronary arteries become more or completely occluded

- ❖ MRI can identify wall thinning scar, scar, delayed enhancement (infarction), and wall motion abnormalities (ischemia). Currently, this is not a primary diagnostic modality for MI, but coronary artery assessment may be enhanced by magnetic resonance angiography (MRA) in the future.

## **MANAGEMENT STRATEGIES**

### **Prehospital care**

The overall goal is to reduce mortality and morbidity of acute Myocardial Infarction. A means to end this is to reduce the time interval from the onset of symptoms to the provision of resuscitation skills, adequate analgesia, adequate assessment and diagnosis and where appropriate, early thrombolytic therapy<sup>80,81</sup>.

## **Strategies**

- ❖ Educating patients regarding recognition of symptoms and call for help.
- ❖ Role of paramedics in recognizing and treating life threatening arrhythmias
- ❖ Integrated response by general practitioners, ambulance service and hospital staff is required.
- ❖ Prehospital thrombolysis - Alteplase for this purpose.

## **Initial Management**

- ❖ Initiation of intravenous line and administration of 5 % Dexrose / saline to keep the vein opened
- ❖ Continous monitoring of cardiac rhythm, heart rate, blood pressure and other vital signs

## **Oxygen administration**

Hypoxemia is common due to ventilation perfusion mismatch. Oxygen administration has been reported to decrease ST-segment elevation in anterior myocardial infarction. So supplementation of Oxygen (2-4 L/min) for all causes of uncomplicated myocardial infarction with  $\text{SaO}_2$  greater than 90% is justified for 2-3 hours. Oxygen administered should be

extended for patients with pulmonary congestion and desaturation<sup>83</sup>.

### **Analgesia**

- ❖ The pain and anxiety contribute to the over activity of autonomic nervous system which increases the myocardial oxygen demand.
- ❖ Morphine is the drug of choice. It is given at a dose of 2-4 mg intravenously at intervals of 5-15 minutes, until pain is relieved.
- ❖ Caution is needed in inferior infarct complicated by right ventricular myocardial infarction<sup>84</sup>, as it produces severe bradycardia due to vagotonic effect.
- ❖ Morphine allays anxiety, relieves pain, causes vasodilation and therefore reduces preload.
- ❖ Pethidine is preferable in patients with inferior wall and right ventricular myocardial infarction.

### **Nitrates ( Sublingual)**

- ❖ Decreases myocardial oxygen demand and increases myocardial oxygen supply. (dilate infarct related vessels)

- ❖ Sublingual nitroglycerine up to 0.4 mg, 3 doses can be administered at about 5 minutes intervals<sup>84</sup>.
- ❖ Should be avoided in the right ventricular myocardial infarction.

### **Beta blockers**

- ❖ Relieves pain by diminishing myocardial oxygen demand
- ❖ Reduces frequency of progression of threatened infarction to complete infarction. It also reduces the infarct size
- ❖ Reduces life threatening arrhythmias
- ❖ Should be initiated within first 3 hours of infarction

Metoprolol is used in the dose of 5 mg intravenously every 2-5 minutes for total of 3 doses. 15 minutes after the last intravenous dose, oral dose of 50 mg every 6 hours for 48 hours, then 100 mg every 12 hours<sup>82</sup> is given.

### **Aspirin**

The use of non enteric coated chewable Aspirin 160-325 mg taken at the onset of symptoms causes 25 % decrease in the mortality. It rapidly inhibits cyclo-oxygenase irreversibly in platelets, followed by reduction of Thromboxane A<sub>2</sub> formation, a mediator of platelet aggregation<sup>84</sup>.

In patients with aspirin allergy or contraindication, Clopidogrel in the dose of 300 mg to 600 mg stratum dose is recommend.

Recent data from the CLARITY trial<sup>85</sup> (Clopidogrel as Adjunctive Reperfusion Therapy Thrombolysis in Myocardial Infarction [TIMI] 28) suggest that adding Clopidogrel to this Aspirin and thrombolysis regimen is safe and effective. The Clopidogrel Dose used was 300 mg. Further studies suggest that a higher dose of Clopidogrel may have added benefit.

## **Reperfusion in Myocardial Infarction**

When ST segment elevation of atleast 2mm in two contiguous precordial leads and 1mm in two adjacent limb leads is present, patient should be considered for reperfusion therapy<sup>84</sup>

### **Primary Percutaneous coronary intervention**

- ❖ Primary Percutaneous coronary intervention is effective in restoring perfusion in STEMI when carried out on an emergency basis in the first few hours of MI<sup>84</sup>.
- ❖ It is more effective in restoring patency of occluded coronary arteries better short term and long term clinical outcomes
- ❖ Primary PCI is preferred when the diagnosis is in doubt, cardiogenic shock is present, bleeding risk is increased or



symptoms have been present for at least 2-3 hours when the clot is more mature and less easily lysed by fibrinolytic agents.

## **Thrombolysis**

Thrombolytic therapy reduces the relative risk of in-hospital death by 50% when given in the first hour of onset of symptoms.

Other uses

- ❖ Limits infarct size
- ❖ Limits LV dysfunction
- ❖ Reduce the incidence of serious complications as septal rupture, cardiogenic shock & malignant arrhythmias

Benefit of thrombolysis possible up to 12 hours, especially if chest discomfort is still present and ST segment remain elevated in ECG.

Goal of thrombolysis is to achieve TIMI-grade 3, flow in the infarct related artery.

### Drugs used in thrombolysis

Drug	Dose
Streptokinase	1.5 million units over period of 1 hour intravenously
Recombinant tissue Plasminogen activator ( rt-PA)	Initial bolus of 15 mg, followed by an infusion of 50 mg or 0.75 mg per kg body weight over next 30 minutes and 30 mg or 0.5 mg/kg over subsequent 60 minutes
Reteplase ( r-PA)	Initial bolus of 15 Mega units followed by a second bolus of 15 Mega units in 30 minutes
Tenecteplase (TNK-tPA)	Direct 0.5 mg/kg bolus

Unless contra indicated, thrombolytic therapy should be given ideally within 3 hours, preferably within 6 hours and can be justifiably given up to 12 hours.

Rt-PA is more effective than streptokinase in restoring the perfusion ( TIMI- grade 3 flow). As bolus fibrinolysis has the advantage of ease of administration, a lower chance of medication error and less non cerebral bleeding , bolus fibrinolytics like reteplase or tenecteplase can be preferred over t-PA.

## **Criteria for thrombolysis**

- ❖ Chest pain consistent with angina
- ❖ ECG changes
  - ST ↑ greater than 1 mm in 2 or more contiguous limb leads
  - ST ↑ greater than in 2 or more contiguous precordial leads
  - New onset LBBB
- ❖ Absence of contraindications

## **Alternative reperfusion regimens<sup>83</sup>**

- Combined intravenous glycoprotein II b /IIIa inhibitor with a reduced dose of fibrinolytic agent
- Facilitated PCI : Glycoprotein II b /IIIa inhibitors followed by PCI
- Pharmacoinvasive approach : Pharmacologic reperfusion regimen followed by routine angiography and PCI after a delay of 12 - 24 hours
- Rescue PCI if failure of reperfusion beyond 90 minutes of thrombolysis
- Elective PCI in coronary artery re-occlusion or recurrent ischemia.

## **Antithrombotic agents**

### **Drugs**

#### **1. Unfractionated heparin**

Immediate administration of Unfractionated heparin helps to facilitate thrombolysis and establish the patency of infarct related artery in t-PA regimen of thrombolysis. It is also gives mortality benefit when added to aspirin and streptokinase regimen.

Dose:

- Bolus 60 u/kg
- Maintenance of 12 U/ Kg / hr
- To keep aPTT – 1.5 to 2 times the control value

#### **2. Low molecular weight heparins**

They have several advantages over unfractionated heparin due to increased anti- factor Xa : IIa ratio, decreased sensitivity to platelet factor IV, as more stable and reliable anticoagulant effect and enhanced bio-availability. There is no need for aPTT monitoring.

Drugs used

Nadroparin	}	efficacy similar to unfractionated heparin
Deltaparin		

Enoxaparin - superior to unfractionated heparin  
( enoxaparin 1 mg/kg subcutaneously every 12 hours)

### **3. Platelet Glycoprotein IIb/IIIa receptor inhibitors**

These drugs facilitate thrombolysis and reduce the rate of reocclusion of reperfused vessels. They act by inhibiting the platelet glycoprotein IIb/IIIa receptors of platelets surface which are the final common pathway in platelet aggregation

#### **Drugs**

- Abciximab is a monoclonal antibody to the IIb/IIIa receptor. It is given in the dose of 0.25 mg/kg intravenous bolus followed by 10 mcg/min over 12 hours
- Orbofiban, sibrafiban
- Tirofiban
- Eptifibatide
- These drugs are more useful in NSTEMI

**4. Bivalirudin** (a direct thrombin inhibitor) has shown some promise in the setting of STEMI if combined with high-dose clopidogrel load and may be an appropriate alternative strategy.

### ***Other drugs***

1. **ACE inhibitors** : These should be prescribed within 24 hours of AMI to all patients with myocardial infarction and overt congestive cardiac failure. It reduces the ventricular remodeling after infarction thereby reduces the subsequent development of failure
2. **Statins** : Statin therapy should be initiated prior to hospital discharge, and perhaps as early as possible, in all patients with an STEMI.

High dose Atorvastatin 80 mg/day used in the PROVE IT-TIMI 22 and MIRACL trials<sup>86</sup> was found effective.

### **Special efforts in the management of Right Ventricular Myocardial Infarction**

The importance of diagnosing right ventricular myocardial infarction lies in its management. The drugs routinely used in treatment of myocardial infarction like sedatives analgesics, anticoagulants, antiplatelet drugs, thrombolytics etc can be given.

However because of the unique hemodynamic features of right ventricular myocardial infarction the use of diuretics and nitrates may precipitate an impending or worsen a preexisting low output state.

## **Volume expansion**

Since there is an imbalance between right and left ventricular filling pressures volume expansion has been advocated to improve the low output state in right ventricular infarction<sup>87</sup>.

Volume expansion should be continued until hypotension is corrected (or) till the pulmonary capillary wedge pressure reaches 15-20 mm of Hg (or) till right atrial pressure reaches the a level of atleast 14-15 mm of Hg.

If central hemodynamic monitoring is not available, one to two liters of saline can be infused while closely following the blood pressure and urine output and examining the patient for signs of pulmonary congestion. If volume loading does not improve hemodynamics or is not tolerated, inotropic therapy should be tried.

But if the left ventricle is also extensively damaged volume expansion will be ineffective. Right atrial pacing has been advocated as an alternative to volume loading.

## **Drugs**

Inotropic agents are indicated when hypo perfusion persists despite adequate left ventricular filling pressure.

Dopamine and Dobutamine may increase the cardiac index and ejection fraction<sup>87</sup>.

When left ventricular failure is present arterial vaso dilators are added to this regimen. They decrease the impedance to left ventricular outflow. Consequently left ventricular diastolic, left atrial and pulmonary arterial pressure also decrease.

This lowers the impedance to right ventricular outflow and enhance right ventricular output. Intra aortic balloon counter pulsation has been used in a few occasions. But it is not clear whether it is useful in a patient with dominant right ventricular infarction.

### **Management of Brady arrhythmias:**

Bradyarrhythmias can be corrected by vagolytics. Temporary pacing may be necessary in refractory cases. When pacing is done, atrioventricular sequential pacing is done as this improves right sided forward flow. Replacement of tricuspid valve has been carried out in treatment of severe tricuspid regurgitation secondary to right ventricular myocardial infarction<sup>88</sup>.

### **Complications of Myocardial Infarction**

#### **Mechanical Complications**

- Ventricular dysfunction
- Cardiogenic shock



- Mitral regurgitation
- Ventricular septal rupture
- Free wall rupture – (cardiorrhesis )

## **Electrical Complications**

### **Tachyarrhythmias**

- These include ventricular premature beats, ventricular tachycardia, ventricular fibrillation and accelerated idioventricular rhythm.
- Supraventricular arrhythmias –sinus tachycardia is the common one. Other common arrhythmias are atrial fibrillation, atrial flutter and paroxysmal supraventricular tachycardia.
- Accelerated junctional rhythm is more common in inferoposterior myocardial infarction

### **Bradyarrhythmias**

- Sinus bradycardia is more common with inferoposterior & right ventricular myocardial infarction managed by atropine. Persistent bradycardia may need pacing.
- AV blocks are particularly common with inferior infarct, attributed to ischemia and enhanced vagal activity and the release of adenosine

- First degree block needs no specific management
- Second degree block particularly high grade type II blocks may require pacing.
- Complete heart blocks are common in inferior infarct and they are transient and get corrected with reperfusion.
- Intra ventricular conduction abnormalities of which LBBB is more common.

### **Specific Complications Of Right Ventricular Myocardial Infarction**

1. Hypotension
2. Conduction defects
3. Thromboembolic phenomenon with a special predilection for pulmonary embolism
4. Severe tricuspid insufficiency
5. Pericarditis
6. Chronic right heart failure in survivors<sup>88</sup>
7. Rupture of Ventricular free wall and interventricular septum<sup>89</sup>
8. Right to left shunting through a patent foramen ovale<sup>90,91</sup>
9. Right ventricular aneurysm<sup>92</sup>

## **AIM OF THE STUDY**

- ❖ To study the various Clinical manifestation of Right Ventricular Myocardial Infarction.
- ❖ To study the Clinical profile, Risk factors, ECG features, Complications and Outcome of Right Ventricular Myocardial Infarction.

## **MATERIALS AND METHODS**

The study was conducted in the Intensive Coronary Care Unit of Government General Hospital, Chennai. The study period was from April 2008 to September 2008.

Design of the study : Prospective Study

Ethical clearance : Obtained

This study was mainly conducted to find out the clinical manifestations of right ventricular myocardial infarction and its risk factors, ECG features, complications and outcome of RVMI.

- All patients admitted in Intensive Coronary Care Unit with ST elevation in V4R were taken for study
- Both male and female patients were included
- Both young and old patients were included

### **Exclusion Criteria :**

- Patients with other congenital and acquired valvular heart disease, COPD and Right ventricular hypertrophy.
- Patients with previous myocardial infarction
- Patients with other serious comorbid illness

## **Study protocol**

### **1. Clinical**

### **2. Electrocardiogram**

## **Clinical Study**

In each case detailed history about age, sex, occupation, presenting symptoms, time of admission, previous history and habits were taken.

Comprehensive risk factor analysis was made with specific importance to

- Obesity
- Diabetes Mellitus
- Hypertension
- Hyperlipidemia
- Smoking status- classified into

- ❖ Non smoker

- ❖ Ex smoker if discontinued 10 years ago and

- ❖ Current smokers

- Family H/o premature coronary artery disease, defined as sudden cardiac death, acute myocardial infarction, coronary artery disease before the age of 40 years

Detailed physical examination was made including

- Vital signs
- Blood pressure
- JVP
- Kussmaul sign
- Cardiac examination for gallops, murmur, rub, rales
- Clinical evidence of tricuspid regurgitation
- Risk stratification with 'Killips' classification

All patients of acute myocardial infarction were given the following treatment.

- Absolute bed rest
- Intravenous line – 5% dextrose to keep the vein open
- Nasal O<sub>2</sub> by mask or prongs
- Aspirin 160- 325 mg tablet chewed & swallowed
- Narcotic pain reliever –Inj. pethidine 50 mg intramuscularly

- Beta blockers, Nitrates and Diuretics are usually avoided as they may cause Hypotension
- Enalapril was started 6 hrs after beta blockers if there was no hypotension
- Sedatives and stool softeners were routinely administered
- Intravenous fluid – Normal Saline was given if hypotension was present. Initially Normal saline was given at the rate of 100 ml/hr. If hypotension was not controlled with volume expansion inotropic agents like dobutamine and dopamine were added to this regimen.
- Anticoagulant - Unfractionated heparin was started 6 hrs after thrombolysis - dose 5000 u i.v. qid

## **Thrombolysis**

Thrombolysis was done depending on the merits of the patient and therapeutic window, excluding contraindications.

Thrombolysis was done with Streptokinase 1.5 Million units dissolved in 100 ml of Normal Saline administered over a period of 1 hour after cardiac monitoring. Adequacy of thrombolysis was assessed in 90 minutes after thrombolysis based on the following parameters.

1. Control of chest pain
2. ST segment resolution
3. Reperfusion arrhythmias

Patients were continuously monitored for complications like infarct extension, re-infarction, left ventricular dysfunction, cardiogenic shock, mitral regurgitation and ventricular septal rupture and managed accordingly.

Electrical complications were continuously monitored and prompt measures were done to correct the same.

Complete basic investigations were done in all patients. These included,

- Urine - albumin, sugar, deposits.
- Urine ketones ( if diabetic)
- Blood - TC, DC, Hb%, ESR.
- Blood - Glucose, Urea, Creatinine
- Serum Electrolytes
- X ray Chest PA view
- Serial CPK-MB estimation was done on admission and every 12 hours for first 48 hours



- Lipid profile was done in all patients on the day of admission

## **Echocardiography**

All patients were subjected to echocardiography after stabilization or at discharge.

The following parameters were noted.

- Wall motion abnormalities in the form of :
  - ❖ Hypokinesia – reduced systolic movement
  - ❖ Akinesia - absence of systolic movement
  - ❖ Dyskinesia - paradoxical motion
- Presence of aneurysm, mitral regurgitation, left ventricular clot or pericardial effusion were noted. Ejection fraction was determined by M-Mode and 2-D using Simpson's rule.
- This was done for patients benefit, but this data was not included in our study

The data were entered in a proforma and analysis of the data collected was done with simple statistical methods.

The patient outcome at the end of first week was assessed in relation to mortality, morbidity with respect to in-hospital complications like electrical, mechanical, left ventricular dysfunction, mitral regurgitation, etc.

# **OBSERVATION & ANALYSIS**

**Study period:** April 2008 – Sep 2008

## **Prospective Study**

Total of 106 patients were studied

## **Clinical Observation**

### **1.Incidence**

During the study period 474 cases of acute myocardial infarction were admitted and . Out of 474 cases 242 were acute inferior wall myocardial infarction. 106 cases showed evidence of right ventricular myocardial infarction in ECG. All the 106 cases were associated with Inferior wall myocardial infarction .

### **2. Sex**

Out of 106 cases , 88 cases were males and 18 cases were females.

<b>Sex</b>	<b>Number</b>	<b>Percentage(%)</b>
Male	88	83
Female	18	17
Toal	106	100

Table 1. Sex prevalance

### 3. Age

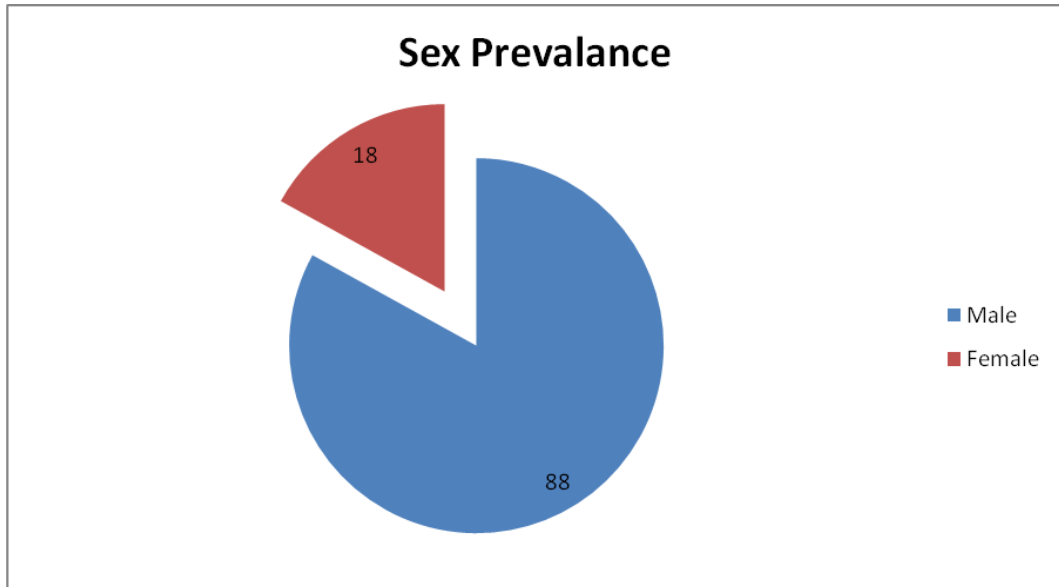
Ages of the patients taken for the study ranging from 26 to 83 years.

- 31 patients were aged between 50-59 years
- The youngest patient in this study was 26 years
- Oldest patient was 83 years old
- In our study highest incidence was in the 6<sup>th</sup> decade

Age	Male	Male (%)	Female	Female (%)	Total	Total (%)
20-29	1	0.9	-	-	1	0.9
30-39	10	9.4	1	0.9	11	10.5
40-49	15	14.2	3	2.8	18	17.0
50-59	31	29.2	8	7.5	39	36.7
60-69	21	19.8	4	3.8	25	23.6
70-79	7	6.6	2	1.9	9	8.5
80-89	3	2.8	-	-	3	2.8
Total	88	83.0	18	17	106	100

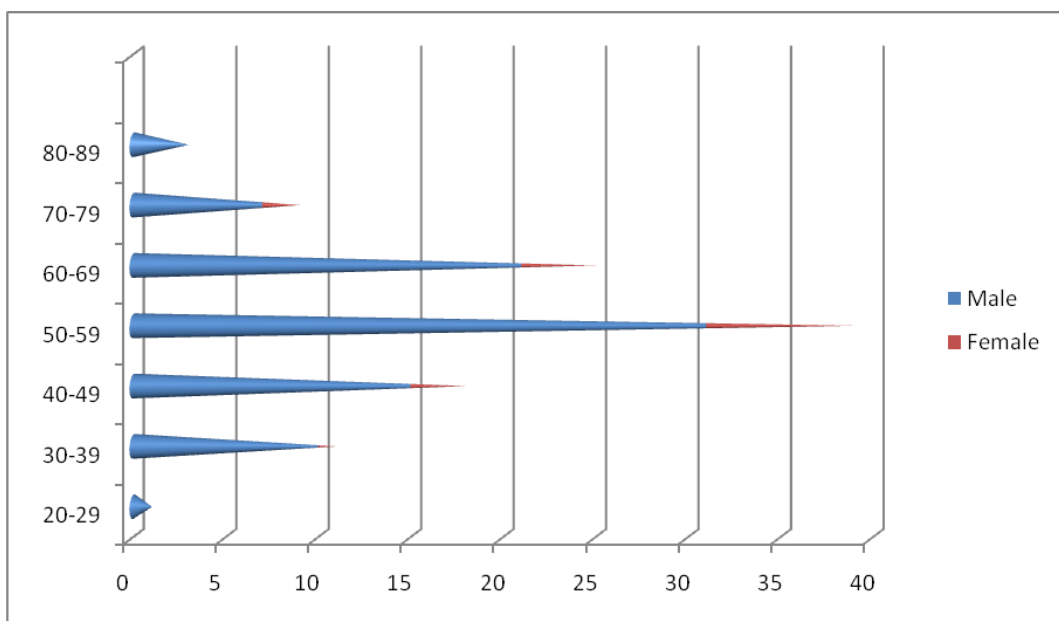
**Table 2. Age prevalence**

**CHART - I**



**CHART – 2**

**AGE PREVALANCE**



#### 4. Occupation

As the occupation of the patient were one of the risk factor, detailed occupational history was analysed in all the patients in this study

Occupation	No. of Patients	Percentage (%)
Professional	7	6.6
Teachers, Clerical staff, Sedentary workers	20	18.8
House wives	16	15.1
Labourers, Coolies	63	59.4
Total	106	100%

**Table 3. Occupation**

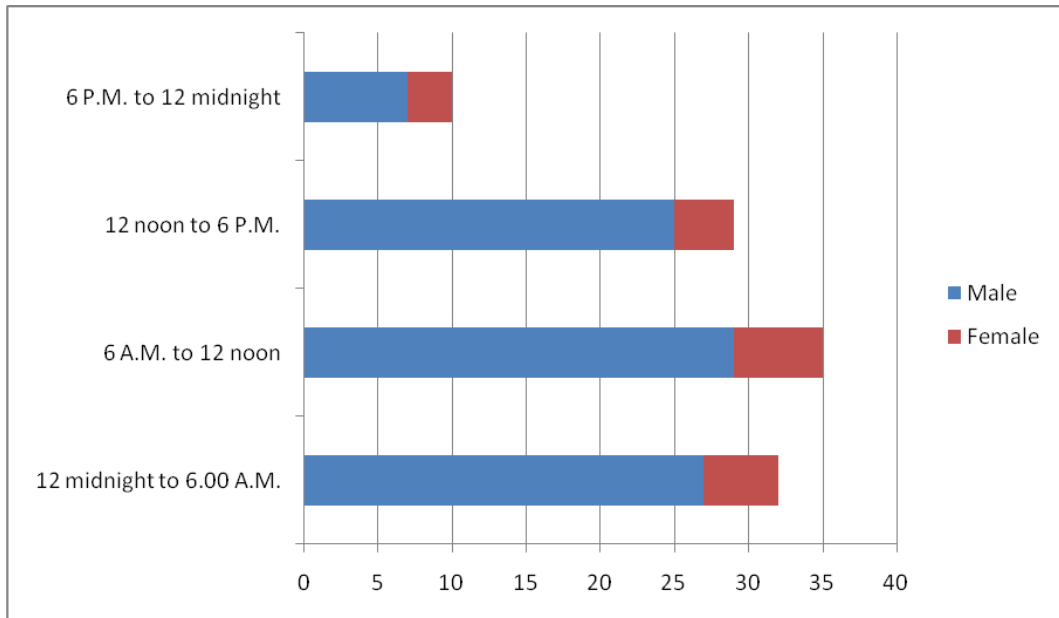
#### 5. Admission time interval after the onset of initial symptom

Time window	Male	Female	Total	Percentage (%)
<3 hour	23	5	28	26.41
3-6 hour	35	7	42	39.62
6-12 hour	20	4	24	22.64
12-24 hour	10	2	12	11.32

**Table 4. Admission time intervals after onset of chestpain**

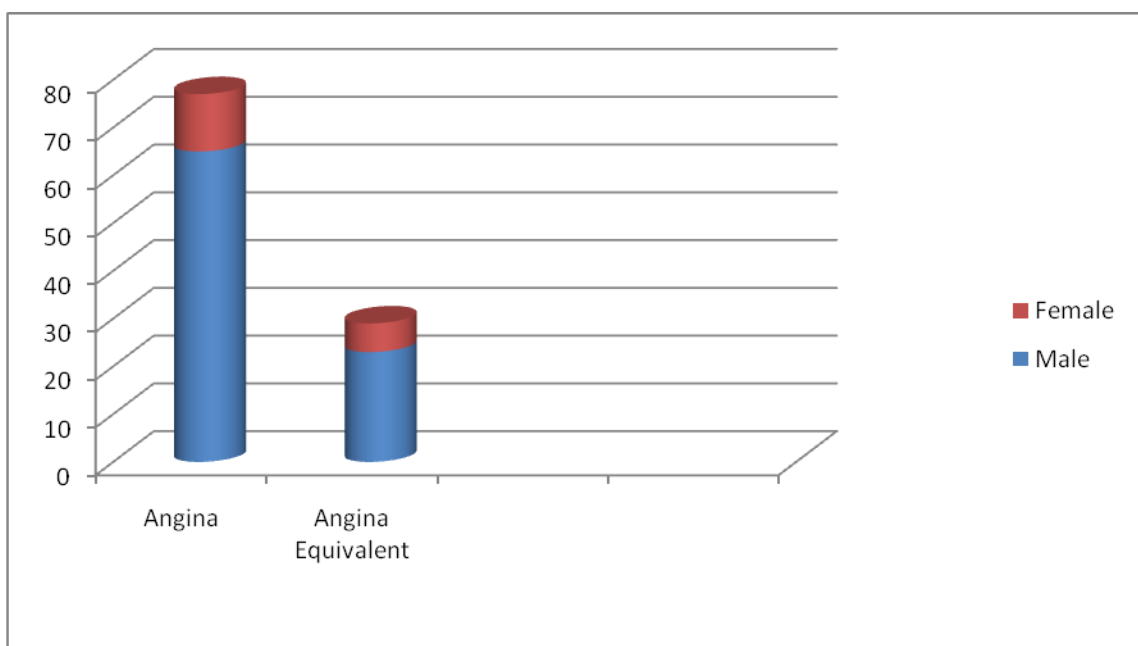
**CHART – 3**

**Diurnal Variation of Symptoms**



**CHART NO. 4**

**Presenting Symptom**



## 6. Diurnal variation of initial symptom

The day was divided into 4 quarters

- Maximum number of patients (29) had their initial symptom in the second quarter between 6A.M.and12noon
- 27 patients had their initial symptoms between 12 midnight to 6 A.M.

Time	Male	Female	Total	Total (%)
12 midnight to 6.00 A.M	27	5	32	30
6 A.M. to 12 noon	29	6	35	33
12 noon to 6 P.M.	25	4	29	27.4
6 P.M. to 12 midnight	7	3	10	9.4

**Table 5. Diurnal variation of Initial symptoms**

## 7. Analysis of Clinical features ( Presenting Symptom)

- Angina was present in 73% of patient
- Angina equivalent in 27 %

Presenting Symptom	Male	Female	Total	Total %
Angina	65	12	77	73
Angina Equivalent	23	6	29	27

**Table 6. Analysis of presenting Symptoms**



- All patients with chest pain were associated with sweating
- Nausea, Vomiting and Indigestion were the common presenting angina equivalents in young
- Dyspnoea was the predominant symptom in elderly people
- Most of the patients with angina equivalents were Diabetes

Gender	Pt with angina equivalents	Dysnoea	Syncope	Giddiness	Nausea/ Vomiting	palpitation	Indigestion
M	23	22	8	16	18	10	6
F	6	5	4	4	5	3	3
Total	29	27	12	20	23	13	9

**Table 7. Analysis of presenting symptoms**

## **8. Risk Factors Analysis**

Risk factors like Diabetes Mellitus, Hypertension, Smoking, Obesity, Family history and Hypercholesterolemia were taken for analysis

- 28 patients had Diabetes Mellitus
- 38 patients were hypertensive
- 53 patients were smokers
- 36 patients had obesity

- 8 patients presented with family h/o Coronary artery Heart Disease

Factor	Male	Female	Total	Total %
Diabetes Mellitus	22	6	28	26.4
Hypertension	31	7	38	36
Smoking	53	-	53	50
Obesity	28	8	36	34
Family History	6	2	8	7.5

**Table 8. Analysis of Risk factors**

## **9. Clinical Signs Suggestive of Right Ventricular Myocardial Infarction**

No	Clinical signs	No. of Cases	Percentage(%)
1	Hypotension SBP < 90	53	50
2	Bradycardia	68	64.25
3	Elevated JVP	55	51.4
4	Kussmaul's Sign	21	19.8
5	Basal Rales	44	41.5
7	Cardiogenic shock	14	13.2

**Table 9. Analysis of Clinical features**

## 10. Risk stratification by Killip Classification

Killip class	No of Cases	Total %	Death
1	51	48.1	
2	28	26.4	
3	16	15.1	
4	11	10.4	7

**Table 10. Killip Classification**

## Electrocardiographic Observations

### 1.ECG Analysis for ST elevation

ST elevation in V4R	No of patients	Percentage(%)	No of Death
1mm	91	85.8	1
>1mm	15	14.2	6
Total	106	100	7

**Table 11. ECG analysis**

### 2. Arrhythmias

- 44% patients developed I degree heart block
- 21% patients developed II degree heart block
- 13.2% patients developed complete heart block
- 3.7% patients developed Atrial Fibrillation

<b>Type</b>	<b>No of cases</b>	<b>Percentage (%)</b>
I degree heart block	46	44
II degree heart block	22	21
Complete heart block	14	13.2
RBBB	3	2.8
LAHB	3	2.8
AF	4	3.7
VF	5	4.7

**Table 12. Analysis of Arrhythmias**

### **Clinical course of the patients after thrombolysis**

#### **1.Improvement of clinical symptoms after thrombolysis**

- Chestpain, syncope, nausea and vomiting improved dramatically with thrombolysis
- 48.1% patients with dyspnoea were also improved with thrombolysis

<b>S.No</b>	<b>Presenting Symptom</b>	<b>No of patients</b>	<b>Symptoms improved in no of patients after thrombolysis</b>	<b>Percentage (%)</b>
1	Chestpain	77	69	89.6
2	Nausea/vomiting	23	23	100
3	Syncope	12	12	100
4	Dyspnoea	27	13	48.1

**Table 13. Improvement of clinical symptoms after thrombolysis**

## 2.Age related mortality ratio

- Out of 106 cases 7 patients died

Age group	No of cases	Death	Percentage of Death
20-29	1	-	-
30-39	11	-	-
40-49	18	1	5.5
50-59	39	1	2.6
60-69	25	3	12
70-79	9	1	11
80-89	3	1	33
Total	106	7	6.6

**Table 14. Age related mortality ratio**

## **DISCUSSION**

A steady decline in the mortality rate from acute myocardial infarction has been observed since 1970. In recent decades, thrombolytic therapy has dramatically reduced the mortality from acute myocardial infarction.

Right ventricular myocardial infarction, usually associated with inferior wall myocardial infarction, with occurrence of complications similar to that in patients with anterior wall myocardial infarction<sup>93</sup>.

So, during early period of right ventricular myocardial infarction we have to carefully watch for the early complications.

The value of conventional ECG as a simple non invasive bed side method for diagnosing right ventricular myocardial infarction has increased recently<sup>4</sup>. Many cases of right ventricular myocardial infarction which were missed during bed side clinical examination were identified by electrocardiography.

## **A. Clinical Analysis**

### **Age and Sex Distribution**

In this study males were affected more than females. This observation usually correlates well with many studies regarding acute right ventricular myocardial infarction<sup>3-10</sup>. This may be due to the risk factors like hypertension, smoking, alcoholism and type A personality which were common in males than females.

### **Age Distribution**

Most of the patients affected were aged between 50-59 years. But in the western studies the highest incidence was in the 7<sup>th</sup> and 8<sup>th</sup> decade<sup>94</sup>

In this study all the females affected were in the older age group than males. This may be due to the protective hormonal effect offered during the pre menopausal period<sup>94</sup>.

### **Occupation**

Various other studies showed that the greater incidence was among those who did sedentary work like executive, businessmen

and other professionals, perhaps some of the rich people may prefer the private sector. But as this study was conducted in Government hospital, greater incidence was noted among the labourers, coolies and weavers.

In this study women after menopause, had increased incidence of myocardial infarction, this is seen especially among house wives who lead a sedentary life.

### **Admission time interval after the onset of initial symptoms and prognosis**

Seven deaths reported in this study occurred in patients who got delayed admission. This delay might be due to poor transport facilities in the village areas particularly at night and also due to atypical chest pain.

### **Inference**

This explains the necessity of integrated health services in the management of acute myocardial infarction and the need for the early transport of the patients to the coronary care units.



## **Diurnal variation**

Maximum number of patients reported their initial symptom in the second quarter of the day between 6 A.M. and 12 noon. It revealed a pronounced circadian periodicity for the time of onset of the acute myocardial infarction with the peak incidence of events between 6 A.M. and 12 noon.

It is supported by **MILIS<sup>95</sup> study – ( MULTI CENTRE INVESTIGATION OF LIMITATION OF INFARCT SIZE)**

## **Reason**

This is due to the early morning rise in the plasma catecholamine and cortisol.

## **Presenting Symptoms**

In right ventricular myocardial infarction nausea with or without vomiting was the presenting symptom irrespective of angina. Angina equivalents were more in females. It was around 40 %. According to Harrison's Principles of Internal Medicine a minimum 15-20 % of Myocardial Infarction are painless<sup>83</sup>. The silent infarcts are greater in patients with diabetes.

Presenting symptoms observed did not vary from various other published studies. As with previous studies prevalence of angina equivalents was 23 % and a higher proportion has been reported in patients with diabetes. Similar observation were made in other studies<sup>96</sup>.

Nausea and vomiting were found to be a common association as given in published observations.

## **Reasons**

In myocardial infarction stimulation of nerve fiber in an ischemic zone of myocardium surrounding the necrotic central area probably give rise to pain. Diabetics with autonomic neuropathy have blunting of this response.

Inferior wall infarction frequently causes increased diaphragmatic and vagal activity which causes vomiting / nausea more frequently.

## **Risk factors**

**Garg et al<sup>97</sup> and Croft et al<sup>98</sup>** observed Diabetes, hypertension and smoking as major risk factors for Myocardial infarction. This studies findings are also consistent with them.

40 % of patients admitted with inferior wall and right ventricular myocardial infarction were found to be hypertensive, equal incidence in males and females. Smoking was exclusively prevalent among males with a frequency of 50 %. Central abdominal obesity, a particular risk factor in India, was found in 36 % of patients with myocardial infarction, more often with females than males in whom it was 60%.

### **Reasons**

The sedentary life style of Indian females, diet habits and lesser inclination to accept modern ways of life styles which involves maintenance of body make up could have contributed this finding.

### **Inferences**

The clinicians should now concentrate, more at identifying those patients who are at increased risk of acute myocardial infarction and benefit from more aggressive prophylactic cardiovascular treatment to prevent it from occurring. More attention should be paid towards prediabetics and prehypertensives to prevent the acute myocardial infarction.

**Presenting signs:**

Hypotension with bradycardia was present in 60 % of patients, a similar clinical observation in other studies. They were transient in many patients. This is due to the activity of Bezold Jarish reflex.

**Cohn**<sup>3</sup> and **Cintron**<sup>99</sup> reported a, 70-100 % incidence of hypotension. **Croft**<sup>98</sup> et al and **Garg et al**<sup>97</sup> reported 33.8 % and 8% incidence of hypotension in patients with and without right ventricular myocardial infarction.

So, whenever a patient with inferior wall myocardial infarction presents with hypotension a co-existent right ventricular myocardial infarction should be suspected.

Systemic venous congestion and raised jugular venous pressure were present in 50 % of the patients. And also a significant number of patients had Kussmaul's sign, oliguria and right ventricular S<sub>3</sub>.

The features of right ventricular failure noted in right ventricular myocardial infarction patients in this study are marginally lower than those reported by **cohn**<sup>3</sup> et al.

**Cintron**<sup>99</sup> reported incidence of right ventricular failure almost similar to that observed in this study.

However in the studies by **wacker**<sup>10</sup> **et al** and **Braat**<sup>100</sup> **et al** none of the patients with right ventricular myocardial infarction, had clinical features of right ventricular failure. So the clinical features of right ventricular failure need not be present in all cases and are present only in cases with severe right ventricular involvement.

Though hypotension with clear lung fields suggestive of right ventricular myocardial infarction, significant number of patients with right ventricular myocardial infarction had basal rales. This probably was due to the coexistent left ventricular inferior wall myocardial infarction in all these patients.

## **B. Electrocardiographic analysis**

**Gupta**<sup>11</sup> **et al** and **Morgera**<sup>13</sup> **et al** proposed ST elevation more than 0.1 mV in the right precordial leads especially in V4R is 100% specific for the diagnosis of right ventricular myocardial infarction. Based on that observation, in this study only those who had ST elevation in V4R were taken for observations.

In a setting of inferior wall myocardial infarction ST elevation in V4R is 100% specific for the diagnosis of right ventricular myocardial infarction.

**Braat et al<sup>100</sup>** and **Kleinho et al** in circulation 1983 reported that ST elevation in right precordial leads is transient. However in this study ST elevation was persistent upto 48 hours after initial ECG in more than 10 % of patients.

This was similar to **Jha<sup>14</sup> et al** and **croft et al<sup>98</sup>** who reported that ST elevation may persist for longer duration upto 72 hours.

ST elevation in right precordial leads indicates right ventricular myocardial damage exceeding 25 % or damage reaching the lateral margins of the right ventricle. So, ST elevation in V4R also indicates extent of the right ventricular damage.

In this study, the lesser the magnitude of ST elevation, lesser the complication was noted. Death rate was observed in patients who had ST elevation more than 1 mm. This shows that the severity of the infarction can be assessed by the amount of ST elevation.

The serious complications like cardiogenic shock, arrhythmias were observed in patients with ST elevation more than 1 mm in this study. This correlates well with other studies<sup>99</sup> also.

**Bradycardia:** Bradyarrhythmias are the commonest electrical complication noted in this study. This is due to stimulation of cardiac vagal afferent receptors.

In this study bradycardia was noted in 2/3 of patients. **Braat et al**<sup>100</sup> reported the incidence of sinus bradycardia in patients with and without right ventricular infarction to be 10 % and 30 % respectively.

In this study also a statistically significant higher incidence of bradycardia was observed. Most of the patients improved with thrombolytic therapy. Similar observation were made by **Cintron**<sup>99</sup> et al.

**Conduction Defects:** **Victor Lagrand**<sup>45</sup> reported 42 % incidence of atrioventricular conduction disturbances in his patients with right ventricular myocardial infarction while **K. C. Garg**<sup>97</sup> et al reported an incidence of 36.4 %.

In this study the incidence of atrio-ventricular conduction disturbances was 78.3 %. Looking into specific conduction disturbances a lower incidence of complete atrio-ventricular block was observed in this study.

Of the 14 cases with complete heart block, 5 patients expired during hospital stay.

Atrial Fibrillation which was transient was noticed in 4 patients(3.7 %). This might be due to left ventricular dysfunction or atrial ischemia. Reported incidence of atrial fibrillation in AMI is 10-15 %, but it is only one third in those with IWMI, as compared to anterior wall MI<sup>101</sup>.

The incidence of intraventricular conduction defects in this study was 5.6 %, i.e., 3 cases had complete RBBB and another 3 cases had left anterior hemi block. The reported incidence of RBBB in AMI is 2-4 % with pure IWMI and 7-10 % with RV extension and that of LAHB is 3.5 % in various studies<sup>100</sup>.



## **C. Clinical course of the patients**

Cardinal Symptoms of Acute Myocardial infarction like angina, nausea, vomiting and breathlessness were improved after thrombolysis.

Early thrombolytic therapy improves the survival rate in patients with acute myocardial infarction. After thrombolysis there was improvement in patients with hypotension and cardiogenic shock.

Out of 106 patients 7 patients died which is comparable with other studies<sup>99</sup>.

## CONCLUSION

1. The incidence of Right Ventricular Myocardial Infarction is fairly common (38%) and its more common in males than females and the difference being less as the age advances.
2. In majority of patients retrosternal chest pain and anginal equivalents like syncope had its onset between 6 A.M. and 12 noon.
3. Smoking and Hypertension were the most prevalent risk factors.
4. All cases of **Inferior Wall Myocardial Infarction should have Right sided chest leads recorded during ECG examination** as more the ST elevation more the severity of Right Ventricular Myocardial Infarction and its complications and mortality rate.
5. If diagnosis of Right Ventricular Myocardial Infarction is correctly made earlier and thrombolysed the prognosis is usually good even in patients with complications.
6. Mortality is higher in patients with Inferior wall Myocardial Infarction with Right Ventricular Myocardial Infarction is because of higher incidence of conduction disturbances and pump failure.

## **SUMMARY**

Right Ventricular Myocardial Infarction is usually associated with Inferior wall Myocardial Infarction, which carries higher mortality and morbidity when it is associated with Inferior wall Myocardial Infarction. Conventional ECG with 12 leads will miss Right Ventricular Myocardial Infarction. So 18 lead ECG is essential to pick up Right Ventricular Myocardial Infarction and Posterior wall Myocardial Infarction early. Early identification and immediate thrombolysis will improve the clinical course and prognosis of Right Ventricular Myocardial Infarction and also reduces the mortality and morbidity many fold.

# PROFORMA

## A Study on Clinical Manifestations of RVMI

Name :

Age :

Sex :

Occupation :

Address :

Date of admission :

Time of admission :

### Presenting Symptoms

1. Chest pain : Y/N

Time of Onset

2. Angina Equivalents

Dyspnoea   Syncope   Sweating   Palpitation   Giddiness  
Indigestion

Past History :

- DM
- HT
- IHD
- CVA
- PVD
- Dyslipidemia

## Personal History

Diet	Veg / Non Veg
Smoking	Y / N
Alcohol	Y / N
Menstrual History	
Oral contraceptives	Y / N

## Family History

DM  
HT  
IHD  
CVA  
Dyslipidemia

## General Examination :

General Appearance :	Comfortable Anxious Apprehensive
Extremities :	Warm Cold Sweating
Anemia :	Y/N
Cyanosis :	Y/N
Clubbing :	Y/N
Pedal edema :	Y/N

## **Vital Signs:**

Pulse :

Blood Pressure :

Respiratory Rate :

Temperature :

## **System Examination**

### **Cardio Vascular System**

- JVP :
- Kussmaul sign :
- Heart sounds : S1 S2 Normal /  
muffled
- Murmur :
- Pericardial rub :

### **Respiratory System**

- Vesicular Breath Sounds -
  - Crepitations
- |           |                 |   |
|-----------|-----------------|---|
|           | Basal           | - |
|           | Upto mid axilla | - |
|           | Upto one half   | - |
|           | Extensive       | - |
| • Rhonchi |                 | - |
| • Others  |                 | - |

### **Abdomen**

- Bowel sounds Y / N
- Hepatomegaly Y / N

- Splenomegaly Y / N
- Free fluid/ ascites Y / N

### **Central Nervous System**

- Level of sensorium :
- Any focal neurological deficit :

### **Investigations**

1. Urine - Albumin  
Sugar  
Deposits
2. Blood - Hb  
TC  
DC  
ESR
3. Blood - Urea  
Sugar  
Creatinine  
Electrolytes
4. Lipid Profile  
Total cholesterol  
Triglycerides  
LDL  
HDL
5. Serum CPK  
LDH  
SGOT  
SGPT
6. Chest X-ray
7. Electrocardiogram  
15 lead ECG -

### Analysis Of ECG

- Rate
- Rhythm
- ST elevation in
- ST elevation in V4R mm
- Reciprocal ST depression
- Heart Blocks

Repeat ECG after 90 min of Thrombolysis

Course in the hospital:

Improved - Transferred to Ward

If death - Cause of death

No days stayed in ICCU :

Outcome:



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# MASTER CHART

S.No	Age	Sex	Occupation	Complaints	Anginal eq	Pain duration	DM	HT	Smoking	Obesity	Bradycardia	Hypotension	JVP	Basal rates	Killip	ST elevation	I av block	2 av block	3 av block	RBBB/LAHB	AE/VT/VF	Death
1	42	m	p	a	-	1	-	-	+	-	-	-	-	-	1	1	+					
2	57	m	sw		+	16	-	+		+	+	+	+	+	2	1	+			RBBB		
3	52	m	p		+	18	+	+	+	+	+	+	+	+	2	1		+				
4	59	m	lab	a	-	7	-	-	+	-	-	-	-	-	1	1		+				
5	42	f	HW		+	14	+	+	-	+	+	+	+	+	3	2					AF	
6	61	m	sw	a	-	10		+	+	-	+	+	+	-	4	2			+			+
7	36	m	lab		+	2			+	+	+	+	+	+	2	1	+					
8	43	m	lab	a	-	11	-	-	+	+	+	+	+	+	2	1	+					
9	57	m	p	a	-	1	-	+	+	-	+	-	-	-	1	1			+			
10	59	f	hw	a	-	8	-	+	-	+	-	-	-	-	1	1	+					
11	53	m	lab	a	-	2	-	-	+	+	+	+	+	+	3	2			+		AF	
12	26	m	sw	a	-	10	-	-	+	-	+	+	+	+	2	1	+					
13	51	m	lab	-	+	1	+	+	-	+	-	-	-	-	1	1	+					
14	38	m	sw	-	+	2	+	-	+	+	+	+	+	+	3	2	+			LAHB		
15	44	m	lab	a	-	11	-	-	+	-	+	+	+	+	2	1		+				
16	54	m	lab	a	-	10	-	-	-	-	-	-	-	-	1	1	+					

17	62	f	hw	-	+	4	+	-	-	-	-	+	+		4	2			+			+
18	34	m	lab	a	-	1	-	-	-	-	-	-	-	-	1	1	+					
19	71	f	hw	-	+	15	+	+	-	-	+	+	+	+	2	1	+					
20	56	m	lab	-	-	16	+	-	+	+	+	-	-	-	1	1			+			
21	63	m	p	a	-	2	-	+	+	-	+	+	+	+	3	2	+			RBBB		
22	48	m	lab	a	-	1	-	-	+	-	+	+	+	+	2	1		+				
23	57	m	lab	a	-	8	-	+	+	+	+	+	+	+	2	1	+					
24	64	m	sw	-	+	16	+	+	+	+	+	+	+	+	3	2		+			AF	
25	56	m	p	a	-	1	-	-	-	-	+	-	-	-	1	1	+					
26	38	m	lab	a	-	2	-	-	-	-	+	+	+	+	2	1	+					
27	58	m	lab	a	-	4	-	-	+	+	-	-	-	-	1	1	+			LAHB		
28	65	f	hw	-	+	5	+	-	-	+	+	+	+	+	3	1	+					
29	43	m	lab	a	-	2	-	-	+	+	+	+	+	+	3	1			+			
30	59	m	lab	-	+	18	+	+	+	+	+	-	-	-	1	1	+					
31	66	m	lab	a	-	8	-	+	+	-	+	+	+	+	2	1		+				
32	59	m	p	a	-	1	-	-	+	-	-	-	-	-	1	1	+					
33	33	m	sw	a	-	2	-	-	+	-	+	+	+	+	2	1						
34	67	m	lab	-	+	18	+	+	+	-	+	+	+	+	4	2			+		VF	+
35	47	m	lab	a	-	1	-	-	-	-	+	+	+	+	2	1	+					
36	54	m	lab	a	-	8	-	-	-	-	-	-	-	-	1	1	+					
37	72	m	lab	a	-	6	-	+	+	-	-	-	+	-	1	1				RBBB		
38	34	m	lab	a	-	8	-	-	+	-	+	+	+	+	2	1	+					
39	73	f	hw	a	-	8	+	+	-	+	-	-	-	-	1	1						
40	68	m	lab	-	+	15	-	-	+	+	+	+	+	+	3	1	+			LAHB		
41	55	m	sw	a	-	2	-	-	-	-	+	+	+	+	2	1						

42	36	m	lab	a	-	7	-	+	+	-	+	+	+	+	3	1		+				
43	69	m	lab	a	-	7	-	-	-	-	-	-	-	-	1	1	+					
44	52	m	lab	-	+	8	+	-	+	+	-	-	-	-	1	1						
45	63	m	lab	a	-	1	-	-	+	-	-	-	-	-	1	1		+		RBBB		
46	44	m	lab	a	-	5	-	-	+	-	-	-	-	-	1	1	+					
47	54	f	hw	a	-	1	-	+	-	+	-	-	+	+	2	1	+					
48	39	m	lab	a	-	1	-	+	+	+	-	-	-	-	1	1	-	+			AF	
49	61	m	lab	a	-	14	+	+	+	+	-	-	+	+	2	1	+					
50	51	m	lab	a	-	7	-	-	-	-	-	-	-	-	1	1	+					
51	58	f	sw	-	+	5	+	-	+	+	+	+	+	+	3	1	-	+				
52	55	f	sw	a	-	2	-	-	-	-	+	+	+	+	2	1	+					
53	48	m	lab	a	-	7	-	+	-	-	+	+	+	+	4	2			+			
54	64	m	sw	a	-	16	-	-	+	-	+	-	-	-	1	1						
55	36	m	lab	a	-	4	-	-	+	+	+	+	+	+	2	1		+				
56	62	f	hw	-	+	2	+	+	-	+	+	+	+	+	3	1	+					
57	51	m	lab	a	-	8	-	-	-	-	-	-	-	-	1	1	+					
58	42	f	hw	a	-	8	-	+	-	-	-	-	-	-	1	1						
59	65	m	lab	a	-	5	-	-	+	-	+	+	+	+	2	1						
60	54	m	p	a	-	17	+	+	+	+	+	+	+	+	4	2		+			VF	+
61	63	m	lab	-	+	6	-	-	-	-	+	-	-	-	1	1	+					
62	43	m	lab	a	-	8	+	-	-	-	-	-	-	-	1	1						
63	52	m	lab	a	-	2	-	-	+	+	-	-	-	-	1	1			+			
64	74	m	sw	-	+	4	-	-	+	-	+	+	+	+	3	1	+					
65	53	m	lab	a	-	5	-	+	+	-	+	+	+	+	2	1						

66	44	m	lab	a	-	8	-	-	-	-	-	-	-	-	1	1		+				
67	64	m	lab	a	-	6	-	+	+	+	-	-	-	-	1	1	-					
68	53	m	sw	a	-	18	+	-	+	-	-	-	-	-	1	1	+					
69	77	m	lab	a	-	8	+	+	+	+	+	+	+	+	3	1	+					
70	41	m	lab	a	-	4	-	+	+	-	+	-	-	-	1	1	+					
71	68	m	lab	a	-	5	-	-	-	-	-	-	-	-	1	1						
72	55	f	hw	a	+	4	-	-	-	+	-	-	-	+	1	1						
73	54	m	lab	a	-	1	-	-	+	-	+	+	+	+	2	1	+					
74	81	m	sw	a	-	8	+	+	-	+	+	+	+	+	3	1	+					
75	37	m	lab	a	-	6	-	-	-	-	+	+	+	-	4	1		+				
76	65	m	lab	-	+	1	-	-	+	+	-	-	-	-	1	1						
77	75	m	sw	a	-	14	-	+	-	-	+	+	+	-	4	2			+		VF	+
78	78	m	lab	a	-	6	-	-	-	-	+	+	+	+	2	1						
79	66	m	sw	-	+	5	+	+	-	-	-	-	-	-	1	1	+					
80	55	m	lab	a	-	2	-	-	+	-	-	-	-	-	1	1						
81	41	m	lab	a	-	5	-	-	-	-	+	+	+	-	3	1		+				
82	67	m	lab	a	-	3	+	+	-	+	+	-	-	-	1	1						
83	53	m	sw	a	-	4	-	-	+	-	+	+	+	+	2	1	+					
84	82	m	lab	a	-	7	-	-	-	-	+	+	+	-	4	2			+			+
85	65	f	hw	-	+	8	-	+	-	-	-	-	-	-	1	1	+					
86	68	m	lab	a	-	5	-	-	+	+	-	-	-	-	1	1		+				
87	83	m	lab	a	-	1	-	+	-	-	+	+	+	+	3	1						
88	55	m	lab	a	-	9	-	+	-	+	+	+	+	+	2	1	+					
89	42	m	lab	-	+	6	+	-	-	-	+	+	+	-	4	2					VF	
90	63	m	lab	a	-	2	-	-	+	-	-	-	-	-	1	1		+				

91	58	m	lab	a	-	4	-	-	-	-	+	-	-	-	1	1			+			
92	79	m	lab	a	-	5	-	-	-	-	-	-	-	-	1	1	+					
93	55	m	lab	-	+	6	+	-	+	+	-	-	-	-	1	1						
94	64	m	lab	a	-	5	-	+	-	-	+	-	-	-	1	1			+			
95	65	m	lab	a	-	4	+	-	-	-	+	+	+	+	3	1	+					
96	46	f	hw	a	-	4	-	-	-	-	-	-	-	1	1			+				
97	55	m	lab	-	+	5	-	-	+	+	+	+	+	2	2	+						
98	32	f	HW	a	-	4	-	-	-	-	-	-	-	1	1	+						
99	46	m	sw	-	+	8	+	-	+	+	+	+	+	-	4	1					VF	+
100	63	m	lab	a	-	4	-	-	-	-	+	+	+	+	2	1		+				
101	57	f	HW	a	-	5	-	-	-	-	-	-	-	-	1	1	+					
102	52	m	HW	-	+	6	-	+	+	-	+	-	-	-	1	2			+			
103	43	m	lab	a	-	6	+	-	-	-	-	-	-	-	1	1						
104	58	f	HW	a	-	2	-	-	-	-	+	+	+	-	4	1						
105	56	f	HW	a	-	1	-	-	-	-	-	-	-	-	1	1						
106	71	m	HW	-	+	5	+	+	-	-	+	-	-	-	1	2			+			



# **PATIENT CONSENT FORM**

## **STUDY TITLE**

### **A STUDY ON CLINICAL MANIFESTATIONS OF RIGHT VENTRICULAR MYOCARDIAL MANIFESTATIONS**

**Study centre** : Department of Cardiology, Madras Medical College

**Patient's Name** :

**Patient's Age** :

**Identification Number** :

Patient may check (v) these  
boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my complete satisfaction.

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason, without my legal rights being affected.

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethics committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.

I agree to take part in the above study and to comply with the instructions given during the study and to faithfully cooperate with the study team, and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms.

I hereby consent to participate in this study of clinical manifestations of Right Ventricular Myocardial Infarction.

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological and urine examination.

Signature / Thumb Impression \_\_\_\_\_ Place \_\_\_\_\_ Date \_\_\_\_\_

Patient's Name and Address: \_\_\_\_\_

Signature of the Investigator : \_\_\_\_\_ Place \_\_\_\_\_ Date \_\_\_\_\_

Study Investigator's Name : \_\_\_\_\_

## சுய ஒப்புதல் படிவம்

### ஆய்வு செய்யப்படும் தலைப்பு

வலது வென்ட்ரிக் கல் மாரடைப்பின் வெளிப்பாடுகள் ஓர் ஆய்வு.

இடம்

: இருதய பிரிவு,  
அரசு பொது மருத்துவமனை,  
சென்னை

பங்கு பெறுபவரின் பெயர்

:

பங்கு பெறுபவரின் எண்

:

பங்கு பெறுவர் இதனை (✓) குறிக்கவும்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டள்ளது என அறிந்து கொண்டேன்.

☐

நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்தும் கொண்டேன்.

☐

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன்.

☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்கமாட்டேன்.

☐

இந்த ஆய்வில் பங்குகொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாரோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்கிறேன்.

☐

இந்த ஆய்வில் எனக்கு இரத்தம், சிறுநீர், எக்ஸ்ரே, ஸ்கேன் பரிசோதனை செய்துகொள்ள நான் முழுமனதுடன் சம்மதிக்கிறேன்.

☐

பங்கேற்பவரின் கையொப்பம் ..... இடம் ..... தேதி

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

ஆய்வாளரின் கையொப்பம் ..... இடம் ..... தேதி

ஆய்வாளரின் பெயர் .....

